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Editorial Note on Aging Skin

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Editorial

Skin aging is a serious cosmetic problem and is associated with significant changes in the function and structure of the skin. The understanding of the underlying basic science of skin aging is rapidly advancing, anchored around nine fundamental aging characteristics identified in 2013. The evidence for the importance of each aging hallmark to skin aging is discussed here, highlighting the uniquely prominent roles of oxidative damage and the extracellular matrix in photoaging. The current evidence for how proven skin aging therapies target each fundamental hallmark is examined and targets for possible future treatments are addressed.

Skin aging is associated with extensive structural and functional changes in all aspects of the skin and serves as a major risk factor for various pathologies, including atopy, wound healing impairment, infection, and malignancy. The aging of sun-exposed skin, with the face in particular, is a major cosmetic concern in addition to functional concerns, prompting patients to seek cosmetic treatment. While sun-exposed skin aging therapies have been present for decades to mitigate or prevent aging, our understanding of the basic science behind aging is rapidly changing, shedding light on existing treatment mechanisms and discovering new treatment objectives and methods. In this paper, we will address the existing understanding of aging hallmarks as applied to skin aging, explore how current therapies target this underlying biology, and discuss potential therapies found by this evolving information.

With different cell populations and underlying biology, the skin consists of two layers. The epidermis, the outermost layer, is a stratified epithelium which consists primarily of keratinocytes. The innermost layer of the epidermis is made up of three differentiated layers of proliferating basal keratinocytes: stratum spinosum, stratum granulosum and stratum corneum. The outermost layer, the stratum corneum, consists of the lipid-rich matrix of anucleate corneocytes. Epidermal stem cells, distinct populations of which are distributed in the basal layer of the epidermis and in the hair follicle, regenerate the epidermis. The dermis, consisting of a collagen-rich extracellular matrix (ECM) supporting vasculature and adnexal structures, lies below the basal layer of the epidermis. Dermal fibroblasts, terminally differentiated cells of mesenchymal origin, produce this matrix.

Sun-exposed skin aging (photoaging) and sun-protected skin (intrinsic or chronological aging) are distinct processes with molecular mechanisms and common and special manifestations. Elevated xerosis (dry skin), fine rhytides and laxity are commonly associated with intrinsic aging. Photoaging shares these traits, but also shows uneven pigmentation, deeper rhytides, telangiectasia, and increased malignant neoplasm growth rate. Histologically, with thickening of the granular layer and more compact corneal layer, dermal ECM loss of collagen and elastin, as well as increased dermal inflammation, photo aging reveals uneven thinning of the epidermal layer. Increased gene expression of matrix metalloproteinase and decreased gene expression of ECM components, especially collagen and elastin, are associated with such histologic findings. These improvements are seen in various photoaging models, and in many mechanistic and therapeutic studies as markers have been used as well.

Since the 1980s, research into the fundamental biology of aging has evolved steadily. The original research focused on the identification of a single driving mechanism. López-Otín et al., however, proposed a different structure for the study of aging biology in 2013, focusing on nine aging hallmarks, which together lead to age-related functional transition. This marked a shift towards considering aging not as a single process, but as a mixture of biological changes instead. Three types are classified into these hallmarks: primary, antagonistic and integrative. The fundamental changes that initiate aging phenotypes are the primary hallmarks of genomic instability, epigenetic transition, loss of proteostasis and telomere attrition. In response to these alterations, antagonistic hallmarks occur and include nutrient sensing de-regulation, mitochondrial dysfunction, and cellular senescence. Together, these all lead to the integrative hallmarks that most specifically contribute to tissue aging phenotypes - impaired intracellular connectivity and stem cell fatigue.

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